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- ☐ 1: [Deng WG, Saunders MA, Gilroy DW, He XZ, Yeh H, Zhu Y, Shtivelband MI, Ruan KH, Wu KK.](#) Related Articles, Li
Purification and characterization of a cyclooxygenase-2 and angiogenesis suppressing factor produced by human fibroblasts.
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Purification and characterization of recombinant human cyclooxygenase-2.
Arch Biochem Biophys. 1994 Nov 15;315(1):111-8.
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Human cyclooxygenase-2 cDNA.

Hla T, Neilson K.

Department of Molecular Biology, Holland Laboratory, American Red Cross, Rockville MD 20855.

Cyclooxygenase (Cox), also known as prostaglandin (PG) H synthase (EC 1.14.99.1), catalyzes the rate-limiting step in the formation of inflammatory PGs. A major regulator step in PG biosynthesis is at the level of Cox: growth factors, cytokines, and tumor promoters induce Cox activity. We have cloned the second form of the Cox gene (Cox-2) from human umbilical vein endothelial cells (HUVEC). The cDNA encodes a polypeptide of 604 amino acids that is 61% identical to the previously isolated human Cox-1 polypeptide. In vitro translation of the human (h)Cox-2 transcript in rabbit reticulocyte lysates resulted in the synthesis of a 70-kDa protein that is immunoprecipitated by antiserum to ovine Cox. Expression of the hCox-2 open reading frame in Cos-7 monkey kidney cells results in the elaboration of cyclooxygenase activity. hCox-2 cDNA hybridizes to a 4.5-kilobase mRNA species in HUVEC, whereas the hCox-1 cDNA hybridizes to 3- and 5.3-kilobase species. Both Cox-1 and Cox-2 mRNAs are expressed in HUVEC, vascular smooth muscle cells, monocytes, and fibroblasts. Cox-2 mRNA was preferentially induced by phorbol 12-myristate 13-acetate and lipopolysaccharide in human endothelial cells and monocytes. Together, these data demonstrate that the Cox enzyme is encoded by at least two genes that are expressed and differentially regulated in a variety of cell types. High-level induction of the hCox-2 transcript in mesenchymal-derived inflammatory cells suggests a role in inflammatory conditions.

PMID: 1380156 [PubMed - indexed for MEDLINE]

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ACCESSION NUMBER: 1994-46604 DRUGU B P E
TITLE: **Characterization of the mechanism of inhibition of
human cyclooxygenase-2 by
anti-inflammatory drugs.**
AUTHOR: Ouellet M; Percival M D
CORPORATE SOURCE: Merck-Frosst
LOCATION: Kirkland, Quebec, Canada
SOURCE: Can.J.Physiol.Pharmacol. (72, Suppl. 1, 453, 19 1 Ref.
CODEN: CJPPA3 ISSN: 0008-4212
AVAIL. OF DOC.: Merck Frosst Centre for Therapeutic Research, Kirkland, QC
H9R 4P8, Canada.
LANGUAGE: English
DOCUMENT TYPE: Journal
FIELD AVAIL.: AB; LA; CT
FILE SEGMENT: Literature

AB The kinetic mechanism of inhibition of the **purified** form of inducible cyclooxygenase (hCox-2) by several classical NSAIDs and a selective Cox-2 inhibitor was determined. Flurbiprofen, meclofenamate and indometacin were all time-dependent inhibitors of hCox-2. None of the 3 inhibitors had a high degree of selectivity, when compared with hCox-1. NS-398 also had time-dependent inhibition of hCox-2, but was a time-independent inhibitor of hCox-1. The difference in the mechanism of inhibition was reflected in the high degree of selectivity observed for hCox-2 over hCox-1. Results demonstrate that the mechanism of inhibition of hCox-2 by classical NSAIDs is similar to that identified for ovine Cox-1. In addition, it shows the nature of time-dependency of inhibition of hCox-1 and hCox-2 greatly determines the degree of selectivity for 1 isozyme over the other. (conference abstract).